OFFICE OF NAVAL RESEARCH CONTRACT N00014-88-C-0118

TECHNICAL REPORT 91-10

REINFUSION OF SHED BLOOD FOLLOWING PEDIATRIC ORTHOPAEDIC SURGERY

BY

F. BLEVINS, B. SHAW, C.R. VALERI, J. KASSER, G. CRAWFORD, AND J. HALL

NAVAL BLOOD RESEARCH LABORATORY
BOSTON UNIVERSITY SCHOOL OF MEDICINE
615 ALBANY STREET
BOSTON, MA 02118

17 JUNE 1991

Reproduction in whole or in part is permitted for any purpose of the United States Government.

Distribution of this report is unlimited.

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Date Entered)

REPORT DOCUMENTATION	•	READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
NBRL, BUSM 91-10		
4. TITLE (and Subiitle) REINFUSION OF SHED BLOOD FOLLOWING PEDIATRIC ORTHOPAEDIC SURGERY		Technical Report
	·	6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(a)	·	S. CONTRACT OR GRANT NUMBER(s)
Field Blevins, Brian Shaw, C. Ro James Kasser, Glen Crawford, and		N00014-88-C-0118
Naval Blood Research Laborat Boston University School of 615 Albany St., Boston, MA	Medicine	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS
11. CONTROLLING OFFICE NAME AND ADDRESS	•	12. REPORT DATE
Naval Medical Research and I	Development	17 June 1991
Command		13. NUMBER OF PAGES
Bethesda, MD 20814 14. MONITORING AGENCY NAME & ADDRESS(II differen	t trom Controlling Office)	15. SECURITY CLASS. (of this report)
Bureau of Medicine and Surgery Department of the Navy Washington, D.C. 20372		Unclassified
		154, DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report)		

Approved for public release and sale. Distribution unlimited.

- 17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)
- 18. SUPPLEMENTARY NOTES

No. 9: Children's Hospital Medical Center, Boston, MA

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Shed blood Orthopaedic surgery Pediatric surgery Blood

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The reinfusion of an average of 336 ml of nonwashed filtered shed blood in children and adolescents following major orthopaedic surgery proved to be a safe procedure. However, the reinfusion of this volume of nonwashed shed blood did not reduce the amount of banked blood transfused. The use of a system for salvage and reinfusion of nonwashed shed blood postoperatively is recommended as a safe method to minimize the need for homologous transfusion, especially when there is insufficient or no pre-donated autologous blood available.

REINFUSION OF SHED BLOOD FOLLOWING PEDIATRIC ORTHOPAEDIC SURGERY

Field Blevins, Brian Shaw, C. Robert Valeri, James Kasser, Glen Crawford, and John Hall

Children's Hospital Medical Center; Naval Blood Research Laboratory, Boston University School of Medicine, Boston, Massachusetts

INTRODUCTION

The transmission of infectious diseases through the administration of perioperative blood transfusions is a serious concern for both patient and surgeon. This is particularly true in major elective pediatric orthopaedic operations such as spinal fusion in which the requirement for intra and/or post-operative transfusions is expected and routine. The incidence of transmission of non-A non-B hepatitis is 3 to 8%^{2,67} and the risk of transmission of acquired immune deficiency syndrome (AIDS) per unit of banked, homologous blood transfused is 1:36,000 to 1:300,000. 12,13,24,49,75 Moreover, many authorities believe that designated donor homologous blood is no safer and perhaps more risky. 6,71 Thus, in an effort to minimize homologous requirements, autologous blood is being used more often.

Utilization of autologous blood may be considered as an alternative to homologous blood at three specific time frames perioperatively. It is now customary in many centers performing major pediatric spine and hip surgery to collect autologous blood before surgery, thereby significantly reducing the need for homologous transfusions. 14,19,26,33,38,

58,73 However, there are limitations to its use; for instance, patients with pre-existing medical conditions such as congenital or neuromuscular scoliosis or myelodysplasia are often unable to predonate autologous blood.

The intra-operative collection of a patient's red blood cells is another situation in which autologous blood has replaced homologous blood. Although there are contraindications to the use of intraoperative blood salvage, even when the blood is centrifuged and washed, for instance, during cancer surgery and sepsis. Present systems centrifuge and wash intraoperatively collected blood to remove non-cellular constituents such as fibrinogen-fibrin split products, free hemoglobin, and debris. There is still some debate about the effectiveness of washing as a means of preventing coagulopathy, renal failure, and cardiopulmonary compromise associated with intra operative blood salvage (TBS) 30.

Collection and reinfusion of autologous blood may also take place during the postoperative period. Postoperative blood salvage (PBS) represents a simple procedure which utilizes a source of autologous blood that would normally be discarded. However, the same concerns regarding malignancy, sepsis, coagulopathy, renal failure, and cardiopulmonary compromise exist for PBS as for IBS. Postoperative blood salvage has been studied to a much lesser extent than IBS; and although results of the two procedures are often lumped together, the physiologic state of the recipient in the two

situations differs markedly, and may have an impact on how the transfusion is tolerated. Although there are approved commercial reinfusion drainage systems available for use following orthopaedic surgery, the safety and efficacy of these systems have yet to be adequately documented. This prospective clinical and laboratory study was undertaken to investigate the safety and efficacy of reinfusing non-washed filtered shed blood following major spine and hip surgery in pediatric and adolescent patients.

METHODS

PATIENT SELECTION: Twenty-nine patients (29) undergoing major hip or spine surgery at The Children's Hospital in Boston between March and July of 1989 were included in the study. Candidates included patients undergoing spine or hip surgery involving decortication of bone with an estimated post operative drain output of over 200 ml and no known malignancy or coagulopathy. Patients were not actually entered into the study until consent of the senior surgeon was obtained preoperatively. In these patients, a Solcotrans drain reinfusion system was used instead of the usual Hemovac drains.

collection of BLOOD: Drains were placed deep and/or superficial to the fascia before closing all spine, hip, and iliac crest bone graft incisions. Bulb suction or gravity drainage was used until 80cm of Hg wall-suction could be

achieved upon reaching the recovery room. Forty (40) ml of acid-citrate-dextrose anticoagulant was used in the Solcotrans receptacle per 500 ml of blood collected in 11 of the 29 patients, but the ACD anticoagulant was discontinued after the eleventh patient on the advice of the Naval Blood Research Laboratory that it was not needed. ³¹ During the first six hours, the blood was collected in the Solcotrans system postoperatively as recommended by Faris and his associates³¹, after which a standard Hemovac bulb suction was used. The drains were removed on the first or second postoperative day.

REINFUSION: Reinfusion of shed blood was started within six hours of placement of the drain, and the minimum volume considered for reinfusion was 200 ml. When the drainage reached the capacity of the Solcotrans receptacle (500 ml) before the six hour time period, it was immediately transfused. A new Solcotrans receptacle was placed when, based on the rate of drainage noted, it was felt that greater than 200 ml would accumulate before the six hour period.

The blood was filtered through a 170 u pre filter and 40 u Pall screen filter and then transfused intravenously by gravity drainage through standard transfusion tubing. The manufacturer's protocol for the use of the Solcotrans was followed, which included priming of the system and inverting the collection chamber.

made of complete blood count (CBC), prothrombin time (PT), activated partial thromboplastin time (PTT), and platelet count, preoperatively, in the recovery room, and daily as clinically indicated. A 10 ml sample of salvaged blood was drawn from the tubing during transfusion for hematocrit determination and anaerobic and aerobic cultures.

In ten patients reinfused with blood without the acidcitrate-dextrose anticoagulant, blood samples were obtained
less than 1 hour after surgery and prior to reinfusion of the
shed non-washed blood, 1-2 hours post reinfusion, 12-18 hours
post reinfusion, and from the Solcotrans receptacle. These
samples were sent to the Naval Blood Research Laboratory for
analysis: measurements were made of hematocrit, hemoglobin,
white blood cell count, platelet count, plasma free
hemoglobin¹⁵, factor V and VIII clotting proteins^{7,32},
fibrinogen³², D-dimer^{5,54}, antithrombin III¹, plasminogen³⁴,
protein C⁵⁷, C3a des arg²¹, and fat particles less than 9
micron, 9 to 40 microns and greater than 40 microns in
diameter.⁴¹

The measured levels of C3a des arg, plasma hemoglobin, D-dimer, and number of fat particles were compared to expected values. The expected levels for C3a des arg, plasma hemoglobin, and D-dimer were determined from the total amount of the substance infused divided by the plasma volume of the recipient. The plasma volume was estimated as 4% of the total body weight of the recipient. For the expected value of the

fat particles in the blood, the recipient's blood volume was estimated as 7% of the body weight.

CLINICAL: The age, weight, sex, diagnosis, and past medical history of each patient were recorded preoperatively. Drain location and position, as well as estimated intra-and postoperative blood loss were recorded. A note was made as to whether the patient received intra- and postoperative transfusions, and whether the blood product was autologous, homologous or Haemonetics Cell Saver-collected washed red blood cells, or Solcotrans non-washed shed blood. Pre- and post-transfusion vital signs were followed for evidence of a transfusion reaction. When possible, blood gas data was analyzed retrospectively.

CONTROL GROUP: In order to investigate the effectiveness of the use of drains, a control group was established from patients in a separate study. The control group consisted of 49 patients in whom standard Hemovac drains had been used. The diagnoses and procedures in the control group were similar to those in the study group, and the patients in the control group were treated by the same attending surgeons as those in the study group. The two groups are compared with respect to transfusion requirements and laboratory parameters where applicable.

DATA ANALYSIS: The means and standard deviations are reported. Data for the study group and the control group were

compared using T-tests. Results prior to and following transfusion of shed postoperative drainage were compared using analysis of variance. 68

RESULTS

Three patients had Solcotrans drains placed but since the drainage was less than 200 ml, these patients were not reinfused. Thus, twenty six of the twenty nine patients entered into the study were transfused with nonwashed filtered shed blood. The average age of the 29 patients was 15.5 years, and mean weight was 51.0 kg (Table 1). Thirty-one procedures were performed on the 29 patients in the study: 16 spine fusions with autologous iliac crest bone grafting; 5 spine fusions using allograft alone, and 10 osteotomies involving the proximal femur and/or pelvis. The most common diagnosis was adolescent idiopathic scoliosis (10 of the 26 patients).

BLOOD LOSS: The average estimated intraoperative blood loss (based on an estimate from drapes, sponges, and suction totals) was eleven hundred and sixty five ml (1165 ml) (Table 1). The total postoperative drainage averaged 638 ml: 388 ml into the Solcotrans drain during the first six hours, and 250 ml into the Hemovac.

TRANSFUSIONS: An average of 340 ml of Haemonetics Cell Saver blood was transfused intraoperatively into 29 patients (Table 1). Each patient was transfused a total of 2.6 units of banked blood, 1.2 units intraoperatively, and 1.4 units postoperatively. The volume of blood reinfused from the Solcotrans drain in twenty six patients averaged 336 ml with a range of 200 ml to 740 ml (Table 1).

CLINICAL PARAMETERS: One patient required platelets and fresh frozen plasma in addition to red cells for postoperative bleeding. A retrospective chart review of blood gas data recorded on 8 patients 1, 24, and 48 hours after the transfusion of unwashed filtered shed blood showed no clinical evidence of acidosis or arterial oxygen desaturation (Table 2). No transfusion reactions were noted based on monitoring of the patients temperature and vital signs in the recovery room, intensive care unit, and the ward (Tables 2 and 3). No transfusions had to be stopped before completion. All cultures on the salvaged blood were negative.

characterization of Reinfused Blood: The average hematocrit of the salvaged blood was 23 V%. In the ten samples analyzed at the Naval Blood Research Laboratory, the in vitro characteristics of the Solcotrans samples collected at time of reinfusion are shown in Tables 4, 5 and 6. The mean platelet count was 28,000 per mm³, and white cell count was 11,400 per mm³. The plasma hemoglobin was 236 mg%, complement C3a des arg was 9376 ng/ml, the number of fat particles less than 9u was 23,643 particles/ml, and the number 9-40 microns was 24/ml. No fat particles greater than 40

microns were observed. Factor V and VIII clotting protein levels were 10% and 35% of normal; fibrinogen was 37 mg/dl; protein C was 52% of normal; antithrombin III was 40% of normal; plasminogen was 54% of normal; and D-dimer was 205 ug/ml.

IN VIVO PARAMETERS: Hematologic measurements made on the patients' venous blood samples obtained immediately before, and 1 and 12-18 hours after reinfusion of the drained blood are shown in Tables 4, 5, and 6.

There were no changes in the hematocrit value or platelet count during the 12-18 hour posttransfusion period, although the white blood cell count did decrease significantly (p=0.003). Plasma hemoglobin increased from about 29 mg% to about 53 mg%, which was equal to the expected value of 56 mg%. C3a des arg increased from 459 to 877 ng/ml, which was less than the expected value of 1454 ng/ml. Fat particles of less than 9 microns in diameter increased from about 1 to 142 particles per ml in 9 of the 10 patients; this level was less than the expected value of 1523 particles per ml. One patient had 3,464 fat particles that were less than 9 microns in diameter per ml prior to transfusion, and data on this patient were deleted from the analysis. Factor V clotting protein increased significantly (p=0.002) from 42 to 65% of normal, and factor VIII increased significantly (p=0.027) from 81 to 102% of normal. Fibrinogen increased significantly (p=0.001) from 154 to 276 mg/dl. There were no changes in the levels of protein C, plasminogen, or antithrombin III. D-dimer level

increased significantly at 1 hour following transfusion and it returned to baseline 12-18 hours posttransfusion.

comparison with control group: Table 1 shows a comparison between the study group and the control group. The study group (mean age of 15.5 years) was significantly older than the control group (mean age 11.8 years). The study group was heavier (51.0 kg) than the control (45.3 kg), but not significantly so. Spinal fusions were performed on 21 of the 31 patients (68%) in the study group compared with 28 of 49 patients (57%) in the control group. Autogenous iliac crest bone graft was harvested in 18 of the 29 study group patients (62%) and in 22 of the 48 in control group patients (45%). In the study group, 9 patients (31%) had only deep drains, 2 (7%) had only superficial drains, and 18 (62%) had drains both deep and superficial to the fascia. In the control group, 31 (63%) had only deep drains, 1 (2%) had only superficial drains, and

Table 1 shows the estimated and measured blood loss for the two groups. The estimated operative blood loss was 1165 ml in the study group and 818 ml in the control group. The postoperative drainage (recorded from Solcotrans and Hemovac) was 638 ml in the study group versus 501 ml in the control group, not a significant difference in either group (p<.05).

The study group was reinfused a significantly greater volume of washed intraoperatively salvaged blood (Haemonetics Cell Saver) (340 ml) than the control group (206 ml) (Table

1). An average of 2.6 units of banked blood was transfused to each patient in the study group (autologous, designated donor, and homologous) compared to 1.2 units for the control group (significant p<.001) (Table 7). Seventy-seven percent (77)% of the banked blood transfused in the study group was autologous compared with 63% in the control group. In the study group, 19 of the 29 patients (66%) received autologous blood, 2 patients (7%) received designated donor blood, and 3 patients (10%) received homologous blood. For the control group, 19 of the 48 patients (40%) received autologous blood, and 10 (21%) received homologous blood (Table 7).

Hematocrit values were similar for the study and control groups. Admission hematocrits were 37 V% for both groups. Immediate postoperative hematocrits were 31 for the study and 33 for the control group (p<.02). The final hematocrits (3-9 days post operatively) were 31 for the study group, and 30 for the control group.

COMPLICATIONS: Two of the 29 patients in the Solcotrans group developed complications:

One patient with Down's syndrome suffered a deep infection and wound breakdown after undergoing a dial osteotomy. This patient initially had Solcotrans drains placed and was switched to a standard Hemovac suction six hours post operatively. Two-hundred (200) ml were reinfused from the Solcotrans, and an additional 185 ml accumulated in the Hemovac before it was removed on the second day post-

operative. A hematoma formed which became infected and led to wound dehiscence. Coagulation studies were normal. The patient required multiple debridements, intravenous antibiotics, and eventual closure with a local muscle flap.

A second patient with severe spastic quadriplegia, who was 11 years old and weighed 20 kg, bled excessively post operatively. During closure of the T3 to L5 Harri-Luque posterior spinal fusion, a marked increase in bleeding was noted from all sites. The estimated intraoperative blood loss was 500 ml; the postoperative Solcotrans and Hemovac output were 740 and 800 ml. respectively. One unit of homologous blood was given intraoperatively, and four units of homologous banked blood, 2 units of fresh frozen plasma and 740 ml of salvaged blood was given postoperatively. Preoperatively, PT, PTT, and bleeding time were at the upper limits of normal; these measurements were elevated immediately postoperatively (PT 17.0/11.9, PTT 34.6/22.6), and had returned to baseline by the second postoperative day.

COST: At the time of this study, the price of one unit (500 ml) of autologous, homologous, and Solcotrans salvaged blood at the Children's Hospital in Boston was \$105, \$234, and \$138 respectively.

<u>DISCUSSION</u>

The reinfusion of shed blood is relatively new in orthopaedics, although it has been used for years in cardio-

thoracic and vascular surgery. Whether non-washed filtered shed blood obtained from the mediastinum should be collected with an anticoagulant and washed prior to reinfusion is still debated. 17,29,42,63,64,69 Washing blood prior to reinfusion reduces plasma hemoglobin concentrations and removes the anticoagulant, fat particles, microaggregates, and debris. reduces the levels of fibrin-fibrinogen degradation products and D-dimer fragments produced by clotting and lysis of the shed blood, as well as products of platelet activation—and lysis (beta thromboglobulin, thromboxane A2, serotonin, and lactic dehydrogenase), and the products of complement activation. Concerns about transfusing unwashed filtered shed blood postoperatively in non-septic, non-oncologic surgery center on possible problems with coagulopathy, complement activation, renal compromise, and contamination by bacteria, fat particles, and other debris. 10,11,22,47,59,61,62

Clements et al²² reported a 25% incidence of complications including hypotension and hypothermia, and one unexplained death, in a study of 16 adult patients undergoing spine surgery and hip and knee arthroplasties who received an average of 200 ml unwashed, filtered shed blood. These results are in contrast to those reported by Faris et al³¹ who found febrile reactions to be the only adverse reaction to reinfusion of an average of 1.3 units of unwashed, filtered blood in 99 hip and knee arthroplasty patients. Groh et al⁴³ noted no evidence of coagulopathy, thrombocytopenia, or renal dysfunction after an average of 607 ml of unwashed blood was

transfused in 25 knee arthroplasty patients. Gannon35 concluded that the postoperative blood salvage and reinfusion of unwashed blood was safe in 124 hip and knee arthroplasty patients. The differences in reported data in the literature on the safety of reinfused unwashed filtered shed blood and the absence of data on its use in the paediatric and adolescent population, stimulated our study of patients in this age group Our findings that platelet count, plasminogen, protein C, and antithrombin III levels in the patient were not significantly decreased indicate that the quantity of unwashed blood infused did not produce a coagulopathy. The findings that shed blood contained high levels of D-dimer, and C3a, as well as low levels of platelets, fibrinogen, and factors V and VIII clotting proteins, indicated an activation of the clotting, fibrinolytic, and complement systems. When this blood was reinfused, the elevations in circulating D-dimer and C3a at one-hour post-transfusion were lower than the expected values. These levels were normal twelve to eighteen hours post reinfusion, suggesting the absence of systemic disseminated intravascular coagulation or complement activation. Similarly, the plasma hemoglobin concentrations which were elevated in the shed blood samples were no higher than expected following reinfusion, indicating that intravascular hemolysis had not occurred.

The shed blood contained on the order of 23,500 particles that were less than 9 microns in diameter per ml.

One hour post-reinfusion the fat particle level was significantly lower than expected, and by 12 hours had essentially normalized. Although the presence of such particles in reinfused blood is of concern, data indicate that the particles are rapidly cleared from the circulation. Clinical observation and blood gas values showed no significant deterioration in oxygenation following reinfusion.

Clinical and vital sign monitoring, temperature data and blood culture results did not show any evidence of significant contamination in shed blood reinfused within the six-hour collection period used in this study. During monitoring in the RR and ICU, there was no evidence of hypotensive episodes associated with the reinfusion of the unwashed filtered shed blood collected with or without the acid citrate dextrose (ACD) anticoagulant.

Our clinical observations and analyses of blood samples correlated well with those of Faris et al³¹ who studied arthroplasty patients reinfused an average of 453 ml of unwashed filtered shed blood. Faris and associates³¹ noted febrile reactions consisting of shaking chills and fevers of 38.5 degrees C. or greater in 14 of 153 patients. They reported a 2% incidence of febrile reactions in patients who received shed blood less than 6 hours after operation, in contrast to a 22% incidence when the blood was not reinfused for 6-12 hours.

Our findings of all negative cultures at time of reinfusion also agree with those of Faris et al 31 . Decker and

Heeg²⁸ found only two positive cultures out of 225 cultures done during intra- and postoperative reinfusion of shed blood and they concluded that intra- and postoperative autotransfusion of shed blood did not increase the risk of infection. Although we noted that the volume and timing of reinfusion appeared to be important considerations in the reinfusion of unwashed shed blood, there was no evidence that the Solcotrans double filtered open-air system as used in our study carried an increased risk of infection over standard blood transfusion.

The possibility that elevated levels of fibrin split products in unwashed shed blood may produce a coagulopathy remains a concern. 9,18,29,37,51,52,55,70 The adolescents and children in our study showed no evidence of an induced coagulopathy after reinfusion of increased levels of fibrin split products. One explanation may be that the volume of shed blood reinfused was small: the patients received an average of 336 ml of shed blood which is approximately 10% of their blood volume. Faris et al³¹ reinfused 10 to 15 percent of the blood volume of their adult patients and did not observe a coagulopathy. Groh et al⁴³ noted no clinical evidence of coagulopathy in reinfusing an average of 607 ml of unwashed filtered shed blood. We agree that further studies are needed on the amount of unwashed filtered shed blood that can be reinfused without producing a coagulopathy in adult and pediatric patients.

When complement is activated, the anaphylatoxins C3a and C5a are formed. 23 They enhance vascular permeability, induce release of histamine, secretion of lysosomal enzymes, and the production of interleukin and prostaglandin from macrophages. 20, 27, 37, 47, 48 Activation of complement also has been proposed as one etiologic factor in the development of respiratory distress syndrome and multi-organ failure. 44,46,56 Bengtson et al¹⁰ in a study of 18 hip and knee arthroplasty patients noted elevated concentrations of anaphylatoxins and terminal complement complexes in postoperatively salvaged blood and hypothesized that activation of the cascade occurred in the collection system or in the wound, although no clinical or laboratory signs of systemic complement activation were noted. Our results correlate well with those findings. Since complications due to complement activation are related to the peak plasma concentration and the duration of elevated concentrations of anaphylatoxins, studies in which larger volumes of nonwashed filtered shed blood are reinfused are necessary to clarify this area of investigation.

The plasma hemoglobin, D-dimer, and fat levels in the nonwashed filtered shed blood were diluted in the blood volume of the recipient following the reinfusion. The hemolysis observed in the nonwashed filtered shed blood was caused by mechanical damage to erythrocytes by negative pressure suctioning, foaming, and contact with air and artificial surfaces. Complement activation may also produce hemolysis. 11 The plasma hemoglobin level of the shed blood in our study of

236 mg% correlated well with the level of 250 mg% found by Faris et al.³¹ The plasma hemoglobin level of approximately 50 mg% found in both our study and Faris and associates, ³¹ is below the level expected to produce hemoglobinuria in patients with normal haptoglobin levels.⁵³

In our study, the acid citrate dextrose (ACD) anticoagulant was added to the collection chamber for the first 11 patients, but was discontinued for the remaining 18 patients since evidence from other studies became available indicating that it was not required and, in fact, might possibly be harmful in hypothermic patients. No gross clots were noted in the collection chamber or tubing, and no drains clotted off. This observation, together with the high level of D-dimer and low levels of fibrinogen, platelets, and factors V and VIII clotting protein, indicated that the shed blood had clotted and lysed. 11,45,66

Because none of the patients in whom ACD was added to the shed blood were included in the group of 10 patietns studied in detail at the NBRL, we are not able to make a comparative analysis of the quality of the nonwashed shed blood with and without ACD. However, Faris and associates³¹ reported no significant differences in their study, with the exception of significantly reduced factor VIII and protein C levels in blood samples collected with ACD.

A major consideration in the reinfusion of shed blood and, in fact, for any blood transfusion, is the red cell survival. There are no published data on the survival of

human postoperatively salvaged blood, although studies in animals have shown 24 hour posttransfusion survival values of 90% and normal lifespan and oxygen transport function. 36,40 Moreover, red blood cells salvaged from patients during cardiopulmonary bypass and vascular surgical procedures and washed before reinfusion have been found to have in vivo survival values similar to those of fresh blood. 3,4 various studies appear to indicate that the survival and function of intra-operatively collected and clotted-lysed washed red blood cells are similar to those of liquidpreserved cells stored for less than a week, and better than Additional those of cells stored for greater than one week. studies of post-operatively salvaged human red cells are needed to determine if these values are similar to those of intra-operatively salvaged red cells.

EFFICACY: The effectiveness of preoperative autologous donation and intraoperative cell salvaging in orthopedic surgery are well documented. 8,16,25,26,31,33,35,37,39,43,50,58,60,65,72,73,77,78 There are fewer reports on the effectiveness of postoperative blood salvage. 65

In our study, we evaluated the effectiveness of the reinfusion of nonwashed shed blood. Forty-eight (48) patients who underwent similar spine and hip procedures at the same institution by the same surgeons and who had standard medium Hemovac drains placed, were chosen as the control group by Crawford et al (unpublished data) comparing this group to a group in which no drains were placed. The demographics of the

control group and the group receiving Solcotrans blood were similar.

Intraoperative and postoperative blood loss observed in the study group was higher, although not statistically significant, than in the control group, and this may have been due to the fact that the study group had a higher percentage of spine fusions (68% vs. 57%) and autogenous iliac crest bone grafts (62% vs 45%). The average weight was greater in the study group, although not significantly so; when blood—loss was reported per kilogram of body weight, the results were similar.

The study group received more cell saver blood and banked blood intraoperatively and postoperatively, which may have been due in part to the greater blood loss observed in this group. Other factors could have influenced the amount of banked blood transfused beside the hematocrit. Wasman and Goodnough⁷⁶ examined the effect of autologous blood donation on physician transfusion behavior. In addition to concluding that significantly lower hematocrits were tolerated in patients with autologous blood deposits, they found that many autologous blood units were transfused because they were available. Although the hematocrits of our control and study groups were not significantly different, a greater percentage of transfused blood was autologous or designated donor in the control population.

CONCLUSIONS

The reinfusion of an average of 336 ml of nonwashed filtered shed blood in children and adolescents following major orthopaedic surgery proved to be a safe proceudre. However, the reinfusion of this volume of nonwashed shed blood did not reduce the amount of banked blood transfused. The use of a system for salvage and reinfusion of nonwashed shed blood postoperatively is recommended as a safe method to minimize the need for homologous transfusion, especially when there is insufficient or no pre-donated autologous blood available.

TABLE 1

BLOOD LOSS AND BLOOD TRANSFUSED IN PATIENTS REINFUSED WITH SHED ORTHOPEDIC DRAINAGE AND PATIENTS NOT REINFUSED WITH SHED ORTHOPEDIC DRAINAGE

	PATIENTS REINFUSED WITH SHED ORTHOPEDIC DRAINAGE	PATIENTS <u>NOT</u> REINFUSED WITH SHED ORTHOPEDIC DRAINAGE	T TEST p VALUE
AGE (years			
MEÀN	15.5	11.8	<0.01
SD	3.6	6.1	
N	29	49	
WEIGHT (k			
MEAN	51.0	45.3	NS
SD	14.9	26.0	
N	29	49	
BLOOD LO	OSS (ml)		
MEAN	1165	818	NS
MEAN SD	627	1018	
SD N	29	49	
		43	
b. POSTO			
MEAN	638	501	NS
SD	424	467	
N	29	48	
c. TOTAL		•	
MEAN	1832	1395	NS
SD	923	1452	
N	29	48	
REINFUSE a. CELL S	D BLOOD (ml) AVER (INTRAOPERATIVE)		
MEAN	340	206	<0.05
SD	257	291	
N	29	49	
b. SOLCO	TRANS (POSTOPERATIVE)		
MEAN	336	0	
SD	186	0	
N	29	49	
c. TOTAL		,	
MEAN	676	206	<0.001 .
SD	324	291	
N	29	49	

TABLE 2

BLOOD PRESSURE AND ARTERIAL BLOOD GAS LEVELS IN PATIENTS FOLLOWING TRANSFUSION OF SHED ORTHOPEDIC DRAINAGE

	1 HOUR FOL	1 DAY LOWING SURG	2 DAYS ERY
SYSTOLIC BLOOD PRESSURE			
MEAN	120	120	118
SD	12	15	15
N	17	22	15
DIASTOLIC BLOOD PRESSURE			
MEAN	68	69	63
SD	9	15	13
N	17	22	15
ARTERIAL PH			
MEAN	7.38	7.39	7.42
SD	0.03	0.03	0.03
N	7	8	3
ARTERIAL p02 (torr)			
MEAN	129	106	138
SD	54	29	54
N	8	8	3
ARTERIAL pC02 (torr)			
MEAN	38.3	38.1	37.3
SD	4.5	4.7	4.2
N	8	8	8

TABLE 3

TEMPERATURE MEASUREMENTS IN PATIENTS TRANSFUSED WITH SHED ORTHOPEDIC DRAINAGE AND PATIENTS NOT TRANSFUSED WITH SHED ORTHOPEDIC DRAINAGE

TEMPERATURE (C)
1 HOUR 1 DAY 2 DAYS
FOLLOWING SURGERY

PATIENTS TRANSFUSED WITH SHED ORTHOPEDIC DRAINAGE

MEAN	37.2	37.5	37.6
SD	0.8	0.7	0.5
N	17	24	18

PATIENTS NOT TRANSFUSED WITH SHED ORTHOPEDIC DRAINAGE

MEAN	 38.0	38.5
SD	0.7	0.6
N	49	46

TABLE 4

HEMATOLOGIC LEVELS IN ADOLESCENT PATIENTS PRIOR TO AND FOLLOWING TRANSFUSION OF 1 UNIT OF SHED ORTHOPEDIC DRAINAGE

-	PRIOR TO TRANSFUSION (RECOVERY ROOM)	1-2 HOUR FOLLOWING	12-18 HOUR TRANSFUSION	ANALYSIS OF VARIANCE p VALUE	SHED ORTHOPEDIC <u>DRAINAGE</u>
HEMATO	CRIT (V%)				
MEAN	28	29	29	NS	23
SD	3	2	2		5
N	10	10	10		19
RANGE	25-	26-	25 -		13-
	32	32	31		29
HEMOGL	OBIN (g/dl)			•	
MEAN	9.8	10.0	9.7	ns ·	7.1
SD	0.9	0.7	0.6		1.6
N	10	10	10		10
RANGE	8.6-	8.5-	8.2-		4.5-
	11.2	10.9	10.4		9.2
WHITE BI	LOOD CELL COUNT ()	(10 ³ /mm ³)			
MEAN	20.2	16.1	12.7*	0.003	11.4
SD	9.4	4.8	3.5		2.6
N	10	10	10		9
RANGE	10.1-	11.0-	6.9-		7.1-
1421.02	42.0	26.5	20.5		15.5
PLATELE	T COUNT (x10 ³ /mm ³)				
MEAN	210	179	176*	0.040	28
SD	83	57	52		38
N	10	10	10		9
RANGE	86-	97 -	107-		4-
142102	303	289	249		123

^{*}Significantly different from pre-transfusion value

TABLE 5

THE MEASURED AND EXPECTED D-DIMER LEVEL, C3A LEVEL, AND NUMBER OF FAT PARTICLES IN ADOLSCENT PATIENTS PRIOR TO AND FOLLOWING TRANSFUSION OF 1 UNIT OF SHED ORTHOPEDIC DRAINAGE

PRIOR TO TRANSFUSION (RECOVERY ROC		NG TRANSF	-18 HOUR USION	ANALYSIS OF VARIANCE D VALUE	SHED ORTHOPEDIC <u>DRAINAGE</u>	
					•	
D DIMED (malmi)						
D-DIMER (ug/ml)	5 O.t.	10.0	9 *7	0.0001	205	
MEAN 0.9	7.8*	12.3	1.7	0.0001	66	
<i>SD</i> 1.3	3.6	6.6	2.3		10	
N 10	10	_10	10		128-	
RANGE 0-	2.0-	5.7-	0-		256	
4.0	16.0	22.6	8.0		256	
PLASMA HEMOGLOBI	N (ma%)			•		
MEAN 29	53	56	26	NS	236	
SD 13	47	16	18		101	
N 9	9	9	9		9	
RANGE 11-		31-	4-		97-	
50	142	75	51		422	
30	#45	, ,	-			
COMPLEMENT C3a de	es arg (ng/ml)					
MEAN 459	3	1454	487	ns	9376	
<i>SD</i> 282	835	594	352		3276	
N 10	10	9	10		10	
RANGE 133	- 199-	592 -	194-		4876-	
1090	2896	2459	1412		13764	
FAT <9 MICRON (# pa	articles/ml)		•			
MEAN 1	142	1523	21	NS	23643	
SD 1	405	3097	59		56965	
N 9	9	9	9		10	
RANGE 0-	- 0-	4	0-		71-	
4	1286	10120	179		194048	
FAT 9-40 MICRON (# particles/ml)						
MEAN 0	0	3	0		24	
SD 0	0	3	0		36	
N 10	10	10	10		10	
RANGE		0			0-	
1011OD		11			95	
FAT >40 MICRON (# particles/ml)						
MEAN	0 0	0	0		0	
SD	0 0	Ō	Ō		0	
	10 10	10	10		10	
RANGE						

^{*}Significantly different from pre-transfusion value

TABLE 6

PLASMA PROTEIN LEVELS IN ADOLESCENT PATIENTS PRIOR TO AND FOLLOWING TRANSFUSION OF 1 UNIT OF SHED ORTHOPEDIC DRAINAGE

Principle Prin		PRIOR TO TRANSFUSION (RECOVERY ROOM)	1-2 HOUR FOLLOWING	12-18 HOUR TRANSFUSION	ANALYSIS OF VARIANCE p VALUE	SHED ORTHOPEDIC <u>DRAINAGE</u>
MEAN 81 70 102* 0.027 35 SD 31 38 43 24 N 10 10 10 10 RANGE 10- 10- 10- 10- 115 126 154 88 FV CLOTTING PROTEIN (%) MEAN 42 46 65* 0.002 10 SD 21 25 26 0 0 N 10 10 10 10 10 RANGE 10- 10- 10- 10 10 REINOGEN (mg/dl) 86 97 97 76* 0.001 37 36 36 36 37 36 36 36 37 36 36 36 37 36 36 36 37 36	EVIII CI C	TTING PROTFIN (%)				
SD 31 38 43 24 N 10 10 10 10 10 10 10			70	102*	0.027	35
N						
RANGE 10- 10- 10- 154 88						
FV CLOTTING PROTEIN (%) MEAN 42 46 65* 0.002 10 SD 21 25 26 0 N 10 10 10 10 10 RANGE 10- 10- 10- 69 86 97 FIBRINOGEN (mg/dl) MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 10 10 10 RANGE 105- 110- 180- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 74 NS 40 SD 11 17 7 7 12 N 10 10 10 10 10 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 110 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-						
FV CLOTTING PROTEIN (%) MEAN 42 46 65* 0.002 10 SD 21 25 26 0 N 10 10 10 10 10 RANGE 10- 10- 10- 10 69 86 97 FIBRINOGEN (mg/dl) MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 10 10 10 RANGE 105- 110- 180- 20- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 70 74 NS 40 SD 11 17 7 11 10 10 RANGE 51- 34- 68- 33- 89 RANGE 38- 41- 45- 30- 34- 80 PROTEIN C (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 10 RANGE 38- 41- 45- 34- 80 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 17 14 77 N 10 10 10 RANGE 33- 35- 32- 44-	KANGE					
MEAN 42 46 65* 0.002 10 SD 21 25 26 0 N 10 10 10 RANGE 10- 10- 10- 69 86 97 FIBRINOGEN (mg/dl) MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 36 36 36 N 10<						
SD 21 25 26 0 0 N 10 10 10 10 10						10
N					0.002	
RANGE 10- 10- 10- 10- 10- 10- 69 86 97						
FIBRINOGEN (mg/dl) MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 10 10 RANGE 105- 110- 180- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 74 NS 40 SD 11 17 7 7 12 N 10 10 10 10 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-						
FIBRINOGEN (mg/dl) MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 10 10 RANGE 105- 110- 180- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 70 74 NS 40 SD 11 17 7 11 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-	RANGE					
MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 RANGE 105- 110- 180- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 74 NS 40 SD 11 17 7 12 12 N 10 10 10 10 10 10 RANGE 51- 34- 68- 33- 33- 34- 68- 33- 33- 34- 40 10		69	86	97		
MEAN 154 203* 276* 0.001 37 SD 41 61 56 36 N 10 10 10 RANGE 105- 110- 180- 20- 225 300 350 110 ANTITHROMBIN III (%) MEAN 70 70 74 NS 40 SD 11 17 7 12 12 N 10 10 10 10 10 10 RANGE 51- 34- 68- 33- 33- 34- 68- 33- 33- 34- 40 10	FIBRINO	GEN (ma/dl)				
SD			203*	276*	0.001	
N				56		36
## ANGE 105- 225 300 350 350 20-				10		10
ANTITHROMBIN III (%) MEAN 70 70 74 NS 40 SD 11 17 7 112 N 10 10 10 10 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-						20-
MEAN 70 70 74 NS 40 SD 11 17 7 12 N 10 10 10 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-	IULIOD					110
MEAN 70 70 74 NS 40 SD 11 17 7 12 N 10 10 10 10 RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-	ANTITUE	OMBIN III (%)				
SD			70	71	NS	40
N 10 10 10 10 10 10 10					110	
RANGE 51- 34- 68- 33- 89 87 80 74 PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 RANGE 38- 41- 45- 34- BROTEIN C (%) PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-						
### 89 87 80 74 PLASMINOGEN (%) ### 89 87 80 74 PLASMINOGEN (%) ### 80 64 56 NS 54 ### 80 10 10 10 10 ### 80 80 69 13 ### 80 80 69 PROTEIN C (%) ### 80 58 57 NS 52 ### 8D 17 17 14 7 ### N 10 10 10 10 10 ### RANGE 33- 35- 32- 44-	_					
PLASMINOGEN (%) MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-	RANGE					
MEAN 50 64 56 NS 54 SD 20 12 9 13 N 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-		89	67	80		7.3
SD 20 12 9 13 N 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 RANGE 33- 35- 32- 44-	PLASMIN	NOGEN (%)				
N 10 10 10 10 10 10 10 RANGE 38- 41- 45- 34- 80 80 69 76 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 10 10 10 RANGE 33- 35- 32- 44-		• •			NS	
RANGE 38- 80 41- 80 45- 80 34- 69 PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-	SD	20	12	9		
PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 10 RANGE 33- 35- 32- 44-	N	10	10	10		
PROTEIN C (%) MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-	RANGE	38-	41-	45-		
MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-		80	80	69		76
MEAN 59 58 57 NS 52 SD 17 17 14 7 N 10 10 10 10 RANGE 33- 35- 32- 44-	DDOTEIN	J C (%)				
SD 17 17 14 7 N 10 10 10 10 10 RANGE 33- 35- 32- 44-			52	57	NS	52
N 10 10 10 10 10 10 RANGE 33- 35- 32- 44-					110	
RANGE 33- 35- 32- 44-						
10 TO TO THE TOTAL TO THE TOTAL TOTA						
	THINGE					

^{*}Significantly different from pre-transfusion value

TABLE 7

ADDITIONAL BLOOD PRODUCTS TRANSFUSED IN PATIENTS REINFUSED WITH SHED ORTHOPEDIC DRAINAGE AND PATIENTS NOT REINFUSED WITH SHED ORTHOPEDIC DRAINAGE

	PATIENTS REINFUSED WITH SHED ORTHOPEDIC DRAINAGE	PATIENTS <u>NOT</u> REINFUSED WITH SHED ORTHOPEDIC <u>DRAINAGE</u>	T TEST P VALUE
ADDITIONAL	BLOOD PRODUCTS TRANS	FUSED (# units)	
MEAN	2.0	0.8	<0.001
SD SD	1.8	1.0	
N	29	49	
# PATIENTS:		18	
* FAITENIS.	66	37	
b. HOMOLOG	OUS RED BLOOD CELLS		
MEAN	0.4	0.4	NS
SD	1.2	1.0	
N	29	49	
# PATIENTS:	3	9	
%:	10	19	
	TED DONOR RED BLOOD CELLS		
MEAN	0.2	0.0	
${\it SD}$	0.9	0.0	
N	29	48	
# PATIENTS:		0	
% :	7	0	
4 EDECH ED	OZEN PLASMAAUTOLOGOUS	•	
		0.0	
MEAN	0.2	0.0	
SD	0.5	. 48	
N	29		
# PATIENTS		0 0	
%:	10	U	
e. FRESH FR	OZEN PLASMAHOMOLOGOU	S	
MEAN	0.1	0.2	NS
SD	0.4	0.6	
N	29	48	
# PATIENTS		4	
%:	3	8	
CRYSTALLO	D		
MEAN	2372	1755	NS
SD	1137	1842	
N	29	48	
# PATIENTS		48	
# PAITENIS	100	100	
			•
COLLOID (AL		140	NS
MEAN	206	140	No
SD	263	218	
N	29	48	
# PATIENTS		15	
% :	45	31	

REFERENCES

- 1. Abildgaard U, Lie M and Odegard OR: Antithrombin (heparin cofactor) assay with "new" chromogenic substrates (S-2238 and Chromozym TH). Thromb Res 11:549-553, 1977.
- 2. Alter HJ and Prince AM: Transfusion-associated non-A, non-B hepatitis: An assessment of the causative agent and its clinical impact. Transf Med Rev 2:288-293, 1988.
- 3. Ansell JE, Parrilla N, King M, Fournier L, Szymanski I, Doherty P, Vandersalm T and Cutler B: Survival of autotransfused red blood cells recovered from the surgical field during cardiovascular operations. <u>J Thorac Cardiovasc Surg</u> 84:387-391, 1982.
- 4. Ansell JE, Vandersalm T, Stephensen W, Szymanski I and Fournier L: In vivo survival of red blood cells processed by a bubble or membrane oxygenator during cardiopulmonary bypass surgery. Texas Heart J 13:247-251, 1986.
- 5. Arocha-Pinango CL: A comparison of the TRCII and latex particle tests for the titration of Fr antigen. <u>J Clin Path</u> 25:757-761, 1972.

- 6. AuBuchon JP: Autologous transfusion and directed donations: Current controversies and future directions.

 Transf Med Rev 3:290-306, 1989.
- 7. Babson AL and Flanagan ML: Quantitative one-stage assays for factor V and X. Am J Clin Path 64:817-819, 1975.
- 8. Bailey TE Jr and Mahoney OM: The use of banked autologous blood in patients undergoing surgery for spinal deformity. J Bone Joint Surg 69A:329-332, 1987.
- 9. Barnhart MI, Cress DC, Henry RL, and Riddle JM:
 Influence of fibrinogen split products on platelets. Thromb
 Diath Haemorrh 17:78-98, 1967.
- 10. Bengtson J-P, Backman L, Stenqvist O, Heideman M and Bengtsson A: Complement activation and reinfusion of wound drainage blood. Anesthesiology 73:376-380, 1990.
- 11. Bengtsson A and Lisander B: Anaphylatoxin and terminal complement complexes in red cell salvage. Acta Anaesthesiol Scand 34:339-341, 1990.
- 12. Berkman SA and Groopman JE: Transfusion associated AIDS. Transf Med Rev 2:18-28, 1988.

- 13. Bianco C: Protecting the blood supply. Transf Med Rev 3:9-12, 1989.
- 14. Blaise G and Jackmuth R: Preoperative autotransfusion for total hip prosthesis. Acta Anaesthesiol Belg 30:175-182, 1979.
- 15. Blakney GB and Dinwoodie AJ: A spectrophotometric scanning technique for the rapid determination of plasma hemoglobin. Clin Biochem 8:96-102, 1975.
- 16. Bovill DF, Moulton CW, Jackson WS, Jensen JK and Barcellos RW: The efficacy of intraoperative autologous transfusion in major orthopaedic surgery: a regression analysis. Orthopedics 9:1403-1407, 1986.
- 17. Bull BS and Bull MH: Enhancing the safety of intraoperative RBC salvage. <u>J Trauma</u> 29:320-325, 1989.
- 18. Carty MJ, Barr RD, Ouna N: The coagulation and fibrinolytic properties of peritoneal and venous blood in patients with ruptured ectopic pregnancy. <u>J Obstet Gynaec Br</u> 80:701-703, 1973.
- 19. Chambers LA and Kruskall MS: Preoperative autologous blood donation. <u>Transf Med Rev</u> 4:35-46, 1990.

- 20. Chenoweth DE and Hugli TE: Demonstration of specific C5a receptor on intact human polymorphonuclear leukocytes.

 Proc Natl Acad Sci USA 75:3943-3947, 1979.
- 21. Chenoweth DE and Hugli TE: Techniques and significance of C3a and C5a measurement <u>in</u> Future Perspectives in Clinical Laboratory Immunoassays, ed RM Nakamura, Alan R. Liss, Inc., NY 1980.
- 22. Clements DH: Salvage andreinfusion of post-operative wound drainage. American Academy of Orthopaedic Surgeons 57th Annual Meeting, page 269, 1990.
- 23. Cochrane CG and Muller-Eberhard HJ: The derivation of two distinct anaphylatoxin activities from the third and fifth components of human complement. <u>J Exp Med</u> 127:371-386, 1968.
- 24. Cohen ND, Munoz A, Reitz BA, Ness PK, Frazier OH, Yawn DH, Lee H, Blattner W, Donahue JG, Nelson KE, and Polk BF: Transmission of retroviruses by transfusion of screened blood in patients undergoing cardiac surgery. NEJM
 320:1172-1176, 1989.
- 25. Cowell HR: Editorial: Perioperative red blood cell transfusion. <u>J Bone Joint Surg</u> 71A:1-2, 1989.

- 26. Cowell HR and Swickard JW: Autotransfusion in children's orthopaedics. <u>J Bone Joint Surg</u> 56A:908-912, 1974.
- 27. Craddock PR, Hammerschmidt D, White JG, Dalmasso AP and Jacob HS: Complement (C5a)-induced granulocyte aggregation in vitro: A possible mechanism of complement-mediated leukostasis and leukopenia. <u>J Clin Invest</u> 60:260-264, 1977.
- 28. Decker K and Heeg P: Microbiology studies of intraand postoperative autotransfusion in orthopedics. Infusionstherapie 17:43-45, 1990.
- 29. Duncan SE, Edward WH, and Dale WA: Caution regarding autotransfusion. <u>Surgery</u> 76:1024-1030, 1974.
- 30. Dzik WH and Sherburne B: Intraoperative blood salvage: medical controversies. <u>Transf Med Rev Vol IV</u> (3):208-235, 1990.
- 31. Faris PM, Ritter MA, Keating EM, and Valeri CR:
 Unwashed filtered shed blood collected after knee and hip
 arthroplasties: a source of autologous red blood cells. _J
 Bone Joint Surg (in press).
- 32. Feingold HM, Pivacek LE, Melaragno AJ and Valeri CR: Coagulation assays and platelet aggregation patterns in

human, baboon, and canine blood. Am J Vet Res 47:2197-2199, 1986.

- 33. Ferrari GM, Zia S, Fella M, Pedemonte R, Profice A and Gamaleri F: Autotransfusion protocol for patients undergoing elective orthopedic surgery. Minnerva Anestesiol 55:227-230, 1989.
- 34. Friberger P, Knos M, Gustavsson S, Aurell L and Clauson G: Methods for the determination of plasmin, antiplasmin, and plasminogen by means of substrate S-2251. Hemostasis 7:138-145, 1978.
- 35. Gannon DM: Evaluation of the efficacy of postoerpative blood salvage after total joint arthroplasty: a
 prospective randomized trial, 57th Annual Meeting American
 Academy of Orthopaedic Surgeons, p268, 1991.
- 36. Giorgio A, Gray A, and Valeri CR: Survival and function of washed red blood cells recovered from clotted blood stored at 22 C for up to 72 hours. <u>Transfusion</u> 29(7):24S, 1989.
- 37. Goodman MG, Chenoweth DE and Weigle WO: Induction of interleukin I secretion and enhancement of humoral immunity by binding of human C5a to macrophage surface C5a receptors.

 J Exp Med 1156:912-917, 1982.

- 38. Goodnough LT, Shafron D and Marcus RE: The impact of preoperative autologous blood donation on orthopaedic surgical practice. <u>Vox Sang</u> 59:65-69, 1990.
- 39. Goulet JA, Bray TJ, Timmerman LA, Benson DR and Bargar WL: Intraoperative autologous transfusion in orthopaedic patients. J Bone Joint Surg 71A:3-7, 1989.
- 40. Gray A and Valeri CR: Survival and function of washed and non-washed red blood cells in dog shed blood.

 Transfusion 29 (7):24S, 1989.
- 41. Greenspan P, Mayer EP and Fowler SD: Nile red: a selective fluorescent stain for intracellular lipid droplets. <u>J Cell Biol</u> 100:965-973, 1985.
- 42. Griffith LD, Billman GF, Daily PO, Lane TA: Apparent coagulopathy caused by infusion of shed mediastinal blood and its prevention by washing of the infusate. Ann Thorac Surg 47:400-406, 1989.
- 43. Groh GI, Buchert PK, Allen WC: A comparison of transfusion requirements after total knee replacement using the Solcotrans autotransfusion device. <u>AABB/ISBT Joint</u>

 Congress Annual Meeting, 1991, S163.

- 44. Hammerschmidt DE, Weaver LJ, Hudson LD, Craddock PR and Jacob HS: Association of complement activation and elevated plasma-C5a with adult respiratory distress syndrome: Pathophysiological relevance and possible prognostic value.

 Lancet 1:947-949, 1980.
- 45. Hauer JM and Thurer RJ: Controversies in autotransfusion. <u>Vox Sang</u> 46:8-12, 1984.
- 46. Heideman M and Hugli TE: Anaphylatoxin generation in multisystem organ failure. <u>J Trauma</u> 24:1038-1043, 1984.
- 47. Henson PM, Larsen GL, Webster RO, Mitchell BC, Goins AJ and Henson JE: Pulmonary microvascular alterations and injury induced by complement fragments: Synergistic effect of complement activation, neutrophil sequestration and prostaglandins. Ann NY Acad Sci 384:287-300, 1982.
- 48. Hugli TE: Complement anaphylatoxins as plasma mediators, spasmogens and chemotaxins in The Chemistry and Physiology of Human Plasma Proteins. Bing DH, ed., Pergamon Press, New York, 1979, pp 255-280.
- 49. Kleinman S and Secord K: Risk of human immunodeficiency virus (HIV) transmission by anti-HIV

negative blood. Estimates using the lookback methodology. Transfusion 28:499-501, 1988.

- 50. Komatsu F: Autologous blood transfusion in orthopedic and oral surgical patients. <u>Bull Tokyo Med Dent Univ</u> 37:51-58, 1990.
- 51. Kopec M, Budzynski A, Stachurska J, Wegrzynowsicz Z and Kowalski E: Studies on the mechanism of interference by fibrinogen degradation products (FDP) with the platelet function. Role of fibrinogen in the platelet atmosphere.

 Thromb Diath Haemorrh 15:476-490, 1966.
- 52. Larrieu JM, Rigollot C and Marder VJ: Comparative effects of fibrinogen degradation fragments D and E on coagulation. <u>Br J Haematol</u> 22:719-733, 1972.
- 53. Laurell CB, Nyman M: Studies on serum haptoglobin level in hemoglobinuria and its influence on renal excretion of hemoglobin. <u>Blood</u> 12:493-506, 1957.
- 54. Mirshahi M, Soria J, Soria C, Perrot JY and Boucheix C:
 A latex immunoassay of fibrin/fibrinogen degradation
 products in plasma using a monoclonal antibody. Thromb Res
 44:715-728, 1986.

- 55. Moore EE, Dunn EL, Breslich DJ, and Galloway WB: Platelet abnormalities associated with massive autotransfusion. J Trauma 20:1052-1056, 1980.
- 56. Moore FD Jr, Warner KG, Assousa S, Valeri CR, Khuri SF: The effects of complement activation during cardiopulmonary bypass: Attenuation of hypothermia, heparin, and hemodilution. Ann Surg 208:95-103, 1988.
- 57. Nicham F, Guichiaoua JF and Martinoli JL: Functional protein C assay using the specific activator from Agkistrodon c. contortrix venom and the new chromogenic substrate CBS 65:25 (to be published).
- 58. Nelson CL, Nelson RL and Cone J: Blood conservation techniques in orthopaedic surgery. <u>Instr Course Lect</u> 39:425-429, 1990.
- 59. Orr MD: Autologous transfusion: a viable alternative.

 J Cardiothor Anesth 2:7-12, 1988.
- 60. Paly W: Effect of preoperative autologous blood donation, intra-operative and post-operative blood recovery on homologous blood transfusion requirement in cementless total hip arthroplasty: a regression analysis. 57th Annual Meeting American Academy of Orthopaedic Surgeons, p 269, 1991.

- 61. Paravinci D: Intra- and postoperative autotransfusion
 -- a quality analysis. <u>Klin Wochenschr</u> 66(15):29-32, 1988.
- 62. Rebeyka DM: Postoperative autotransfusion: an overview of techniques and clinical efficacy. Can Crit Care Nurs J 7:4-11, 1990.
- 63. Schaff HV, Hauer JM, Bell WR, Gardner TJ, Donahoo JS, Gott VL and Brawley RK: Autotransfusion of shed mediastinal blood after cardiac surgery. A prospective study. <u>J Thorac Cardiovasc Surg</u> 75:632-641, 1978a.
- 64. Schaff HV, Hauer JM and Brawley RK: Autotransfusion in cardiac surgical patients after operation. <u>Surgery</u> 84:713-718, 1978b.
- 65. Semkiw LB, Schurman DJ, Goodman SB and Woolson ST:

 Postoperative blood salvage using the Cell Saver after total
 joint arthroplasty. <u>J Bone Joint Surg</u> 71:823-827, 1989.
- 66. Silva R, Moore EE, Bar-Or D, Galloway WB and Wright ED: The risk:benefit of autotransfusion: Comparison to banked blood in a canine model. <u>J Trauma</u> 24:557-564, 1984.

- 67. Sirchia G, Giovanetti AM, Parravicini A, Bellobuono A, Mozzi F, Pizzi MN and Almini D: Prospective evaluation of posttransfusion hepatitis. <u>Transfusion</u> 31:299-302, 1991.
- 68. Snedecor GW and Cochran WG: Statistical Methods. 6th Ed., Ames, Iowa: Iowa State University Press, 1973.
- 69. Solem JO, Olin C, Tengborn L, Nordin G, Luhrs C, Steen S: Postoperative autotransfusion of concentrated drainage blood in cardiac surgery. Experience with a new autotransfusion system. Scand J Thorac Cardiovasc Surg 21(2):153-157, 1987.
- 70. Stillman RM, Wrezlewicz WW, Stanczewski B, Chapa L, Fox MJ, and Sawyer PN: The haematological hazards of autotransfusion. Br J Surg 63:651-654, 1976.
- 71. Strauss RG and Sacher RA: Directed donations for pediatric patients. <u>Transf Med Rev</u> 2:58-64, 1988.
- 72. Thomson JD, Callaghan JJ, Savory CG, Stanton RP, Pierce RN: Prior deposition of autologous blood in elective orthopaedic surgery. <u>J Bone Joint Surg</u> 69A:320-324, 1987.
- 73. Turner RH, Capozzi JD, Kim A, Anas PP, and Hardman E: Blood conservation in major orthopedic surgery. Clin Orthop 256:299-305, 1990.

- 74. Valeri CR: Physiology of blood transfusion. In: Surgical Intensive Care, eds. GT Shires and PS Barie, Little, Brown and Co., (in press).
- 75. Ward JW, Holmberg SD, Allen JR, Cohn DL, Critchley SE, Kleinman SH, Lenes BA, Ravenholt O, Davis JR, Quinn MG and Jaffe HW: Transmission of human immunodeficiency virus (HIV) by blood transfusions screened as negative for HIV antibody. NEJM 318:473-478, 1988.
- 76. Wasman J and Goodnough LT: Autologous blood donation for elective surgery. <u>JAMA</u> 258:3135-3137, 1987.
- 77. Wilson WJ: Intraoperative autologous transfusion in revision of total hip arthroplasty. <u>J Bone Joint Surg</u>
 71A:8-14, 1989.
- 78. Woolson ST, Marsh JS and Tanner JB: Transfusion of previously deposited autologous blood for patients undergoing hip-replacement surgery. <u>J Bone Joint Surg</u> 69A:325-328, 1987.